THE COURSE OF SENSORY IMPULSES IN THE SPINAL CORD[1]

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Dissociation of sensation occurs in cerebral affections and also in affections of peripheral nerves, but most commonly from affections of the spinal cord. The extent of this last dissociation of sensation is topographically similar to that of complete anesthesia from spinal lesion. It has been described as produced by trauma to the spinal cord, syringomyelia, pachymeningitis (Herzen (1)); traumatic hematomyelia (Minor (2), Bergman (3)); tumor of the cord (Bruns (4) and Schlesinger (5), Cushing (6)); gumma (Lahr (7), Hanot (8)); glioma (Henneberg (9)); spinal syphilis (Brissaud (10)); compression of the cord (Edsall (11), Marinesco (12), Vines (13)); myelitis (Pick (14)); and various other spinal diseases and injuries. It will be seen that the dissociation occurs in widely varied conditions and probably depends on the part of the spinal cord affected rather than on the nature of the process.

At the present day it is generally conceded that various forms of sensation are conveyed upward in the spinal cord by systems of fibers which take their course in different portions of the cord. This view, first announced by Van Gehuchten (15) in 1893, according to his own statement, was at first thought to be true only for fibers conveying sensation of pain and temperature. Since then it has been found possible to separate the course in the spinal cord of other forms of sensation.

This theory superseded the old idea that the losses of different forms of sensation were due to different degrees of destruction of the same fiber tracts, and is also better supported by the clinical and pathological evidence than the hypotheses of those who assert that all forms of sensation are carried interchangeably by the same fibers within the spinal cord, and that their different qualities are due to their different effects on the patient's mind.

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The literature on the subject of the course of the sensory impulses in the spinal cord is very extensive. Long bibliographies are appended to the papers of Piltz (16), Henneberg, and Head (17). Head takes up the subject from a somewhat different aspect than the others, being chiefly concerned with the purely physiological side. As he says, the cases which have been carefully and accurately studied clinically and also anatomically are very rare. He had post-mortem examination in only two of his cases and in these the changes which evidently occurred shortly before death (hemorrhage, etc.) throw some doubt on the relation of the symptoms to the lesion found.

It is hardly necessary to review herein all the theories that have been held by the numerous writers as to the course of the sensory impulses in the spinal cord. I shall present only my conclusions as to the position and relations of these fiber systems in the spinal cord drawn from a study of the literature and also a number of cases in the Hospital of the University of Michigan, in which there was a dissociation of the various forms of sensation due to lesion within the spinal cord. The case histories presented and pathological findings are abstracted to that part which bears directly on the question in hand.

The fibers conveying sense of pain enter the posterior horn from the posterior root and ascend in the gray matter for a variable distance which probably depends on the level in the cord, and which varies from two to eight segments, since in cases of unilateral lesion of the cord with autopsy the lesion is usually found to be a constant distance above the limits of the analgesia of the contralateral side; this distance depending partly on the level of the cord affected and partly on the depth of the lesion; those extending into the gray matter cause the analgesia to extend higher. Pétren, after an analysis of one hundred and seventy-five cases, comes to nearly the same conclusion. The fibers may be interrupted by ganglion cells in the posterior horns. They cross to the contralateral side of the cord and ascend in the column of Gowers, part going to the cerebrum, either directly or indirectly and conveying a conscious perception of pain, and part to the other terminations of Gowers tract, the cerebellum, inferior olive, etc., for the establishment of the pain reflexes.

All of the above conclusion is supported by numerous cases in the literature, except the last part which is a hypothesis based on the anatomical descriptions of the course of the tract of Gowers in combination with the well-known physiological response to pain stimuli. It is often observed that in analgesia from cerebral affection the patient reacts to certain stimuli as if painful, though he says he feels no pain. The cases of Thos. J.; Harry G. from negative standpoint; Ada J.; and Ina W. bear out the above conclusion.

Fibers conveying sensations of heat and cold enter the spinal cord from the posterior roots and follow much the same course as those conveying pain sensation, from which, however, they are distinct and separate. This conclusion is based on the observations that while pain sense is usually lost in the same area as temperature sense from spinal lesion, it is not invariably so. In a number of cases in the literature the upper limit of lost temperature sense from traumatic lesion of the spinal cord does not correspond with the upper limit of analgesia (case of Chas. S.). In some cases of Brown-Séquard paralysis with lost pain and temperature senses the one returns while the other does not (Ada J.) or is lost while the other is not. Finally, sense of cold may be lost while sense of warmth is retained as in Mai's (18) case, or the reverse may occur as in my case (Thos. J.), thus apparently indicating that there are separate fibers for each of these sensations. It would seem probable that the system of fibers conveying temperature sense is more widely separated from that conveying pain sense in the cervical region of the cord and in the medulla than in the lower levels of the cord, as the cases of this form of dissociation are more frequently reported from injury or lesion at these levels (Raymond and Lajonne (19), and others).

Tactile sensation enters the spinal cord by the posterior roots and is conveyed upward in the posterior column on the same side at first, though it probably decussates higher up, as shown by Head's cases. From the fact that pain and temperature senses are quite frequently lost in spinal cord lesions when touch is not affected, it seems probable that there is a considerable separation of these fiber systems (Ada J. and other cases). I infer that touch sensation ascends in the cord without immediate decussasation because of the well-known dissociation of sensation in syringomyelia. In my own series of cases, in those in which there was no anesthesia there was no degeneration of the posterior columns (Thos. J.), and in those in which there was anesthesia there was degeneration in the posterior columns (Margaret B., combined to some extent; and Harry G., tactile anesthesia without analgesia), and this leads me to believe that the posterior columns chiefly, if not alone, convey tactile impressions. Cases reported in the literature confirm this conclusion, notably that of Meyer (20), in which there was a transverse myelitis involving all parts of the cord except the dorsal two thirds of the posterior columns and in which there was complete analgesia and thermanesthesia from the sixth rib on the right and fifth rib on the left on down the body, and in which there was no anesthesia, tactile sense was everywhere preserved.

Sense of position is probably a sensation coming from joints; sense of motion comes from muscles (Case of Chas. S.). In normal individuals probably both position and motion are recognized by the aid of sensations from both sources. Fibers enter the posterior root and the posterior horn and run to the cells in Clarke's column or that vicinity; possibly ascend in the gray matter of the same side for a variable number of segments, depending on the level in the cord; and then continue their course upward in the direct cerebellar tract of the same side. It is possible also that sense of position is conveyed upward partly in the posterior columns, which is a time-honored view based largely, I think, on the loss of the sense of position of the lower extremities which occurs in tabes. Since in tabes, however, the primary trouble appears to be in the posterior roots and ganglia, it is not a fair inference. Ataxia does not occur markedly even when the posterior columns are considerably diseased (Harry G.). That the impulses conveying the sense of motion and position pass upward on the same side of the spinal cord as that in which they enter is abundantly proved by the number of cases of Brown-Séquard paralysis on record in which this sensory loss is on the same side as the paralysis below the lesion (case of Ada J.; also case of Jolly (21) and Eskridge and Rogers (22)).

Case 1.[1a] Ada J., a school girl, aged seventeen years, was admitted to my service in the University Hospital on November

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[1a] Sensation to touch was tested by light strokes with the finger; sensation for pain, by pin pricks; sensation for heat and cold, by the application of test-tubes filled with water at a temperature of approximately 50° and 0° Centigrade.

3, 1907, complaining of numbness of the left leg and an inability to move the right leg. Her family history was negative, as was also her previous medical history. She began menstruating at the age of fifteen years and it was regular and painless. She had been menstruating for one day when her present trouble came on and then it stopped. Eight days previous to her admission to the hospital she went to bed feeling perfectly well; at 4 A. M. the next morning she awoke with a severe pain of a drawing character in the epigastrium. She came downstairs to get a drink of water, then returned to her bed and to sleep, so there was evidently no weakness or ataxia at that time. This pain was present, but not severe in the morning when she arose at the regular hour. Her legs felt weak, but she walked about until noon, when she lay down because her left leg felt numb, and an hour later she found that she could not move her right leg and could not sit up, and she also had some pain in the thoracic region of the spine. Her condition had remained about the same up to the time of her admission to the hospital.

Physical examination on admission showed: height five feet seven inches, weight one hundred and twenty-five pounds. Nutrition good, cheeks slightly flushed, vaccination scar on the left arm. The examination of her heart and lungs was entirely negative. The liver and spleen were not enlarged or tender. The abdomen was negative.

On examination it was found that her visual form field was not contracted. Her pupils were equal, somewhat dilated and reacted promptly to light and in accommodation. The extraocular movements were normal and there was no nystagmus or diplopia. The senses of smell and taste were normal. There was no paralysis in the distribution of the fifth or seventh cranial nerves. She felt touch and pinpoint normally on both sides of the face, on both sides of the upper chest and over both arms, and there was no disturbance of the temperature sense or of the sense of position in these regions. The tongue protruded straight and there was no tremor or atrophy. The pharyngeal reflex was present on both sides. The movements of the neck were of normal strength and there was no rigidity of the neck. All movements of the shoulders, elbows, wrists and fingers were strong and equal on each side. There was no intention tremor or ataxia of the hands. She felt pinpoint better on the skin than on the nipple and touch better on the nipple than on the surrounding skin (Graves' stigma of hysteria was absent). She could not move the right leg at any part and could draw up the pelvis on the right side only slightly. She could elevate the left leg from the bed, but not with much strength, as the weakness of the other leg provided no support. She could flex and extend the knee, ankle and toes on the left side freely and with strength. There was normal tactile sensibility over both legs and the abdomen. Pain sense was lost in the left leg and the left side of the abdomen up to about two inches below the level of the umbilicus. On the outside of the thigh the loss of pain sense extended up to about two inches above the crest of the ilium. On the right side of the abdomen there was a zone of lost pain sense extending from about two inches below the umbilicus up to the level of the ensiform cartilage. These areas of lost pain sense were extended around the back to the spine with the gradual sloping of the line upward, as is seen in these cases of anesthesia from spinal lesion. Temperature sense both to heat and cold was completely lost in the left leg and up to the level of the umbilicus, and it was also lost in a zone about the right side of the body extending from the level of the umbilicus to the level of the ensiform cartilage. There was a loss of the sense of position of the toes, ankle and knee on the right side, but no loss of sense of position in the corresponding joints of the left leg. She could raise herself to a reclining posture on the left elbow, but not on the right. The spine was held somewhat rigid, but there was no deformity and no tenderness. The supraorbital reflexes were present. The biceps jerks were present and about equal on the two sides. The knee jerks were slightly diminished on both sides. The Achilles jerks were present. There was no ankle clonus on either side. There was a normal plantar reflex on the left side with a distinct Babinski reflex on the right side. The abdominal reflex was present on the left side, but absent on the right side.

The urine examination showed a specific gravity of 1025, reaction acid, and was negative for albumin, glucose, bile, acetone, and indican in pathological amount. There was a slight sediment, chiefly of oxalates.

The blood examination showed: hemoglobin 90–100 per cent. (Talquist) ; white blood cells 6400, red blood cells 6,020,000 ; the differential count of five hundred white blood cells showed: polynuclear 51.2 per cent., large lymphocytes 4 per cent., small lymphocytes 28.8 per cent., transitionals 7.6 per cent., esinophiles 8.4 per cent.

The patient was placed in bed and given small doses of potassium iodid and on November 18 it was noted that she could move the toes of the right foot and could move the right ankle slightly. On November 25 it was noted that she could draw up the right leg on the abdomen and could extend it, but not with strength. The right leg was distinctly colder than the left to the touch and had a bluish appearance. The sense of position was still lost in the right knee, ankle and toes, and other sensations showed no change from the previous examination. The right knee jerk and Achilles jerk were exaggerated and there was an ankle clonus on the right side. The knee jerk and Achilles jerk on the left side were normal.

She gradually continued to improve, so that by December 16 she was walking about with only a slight limp on the right side. All movements of the right leg were weaker than those of the left leg. There was no atrophy anywhere in the arms or legs. The right knee jerk was exaggerated and showed a patellar clonus. The right Achilles jerk was exaggerated and showed an ankle clonus. Plantar irritation caused extension of all the toes when performed on the right side, flexion of all the toes on the left side. There was no loss of sense of position of the toes or ankle on either side. Sensation to touch was normal all over the arms, legs and body. The senses of pain and temperature were lost to much the same extent as at the first examination. In the areas of lost temperature sense she was unable to differentiate heat and cold (5° and 50°), and said that she felt both merely as touch. She left the hospital January 15, 1908, in much the same condition as at the examination December 16, 1907.

CASE 2. Chas. S., an engineer, aged forty-three years, was admitted to the University Hospital on December 11, 1907. Chief complaint: spinal injury. His family history and previous medical history were negative. On the evening previous he was in a railroad wreck and jumped from an engine, striking against a car. He was unconscious, but regained consciousness in a few minutes. He had some cuts about the head and face; was paralyzed and anesthetic from the chest down and had three fractured ribs on the right side. He complained of pain in the chest and complete retention of urine.

Examination.—Head was bandaged for various cuts. Had a cut over the bridge of the nose. The left eye was blackened and there was sub-conjunctival ecchymosis. The left palpebral fissure was narrower than the right, but he said this has been so since childhood. The left pupil was a trifle wider than the right. Both pupils reacted promptly to light and in accommodation. There was no ocular palsy. There was no facial paralysis. There was no palsy of the tongue. He felt touch and pinpoint equally well on each side of the face and neck. He did not rotate his head because he said it hurt the back of his neck, but there seemed to be no rigidity of the neck. All movements in both arms were apparently normal, well performed and strong. He did not raise his arms above his head because of the pain in the chest caused thereby. He felt touch and pinpoint equally well in each arm, and all over both arms. Distinguished heat and cold all over both arms. No loss of stereognostic sense. No loss of sense of position in the fingers. Sensation to light touch was normal on the left side down to the lower border of the third rib. At the left border of the sternum the line dipped down to about the level of the upper border of the fifth rib, where it crossed the sternum, rose slightly to the lower border of the fourth rib and followed the lower border of the fourth rib around the chest. Sense of pain to pinprick was normal on both sides down to the upper border of the third rib in the mid-clavicular line and the line was directly horizontal around the chest. Sensation of differentiation between moderate degrees of heat and cold was lost below the same line as the pain sense. Below this line and down to the line of lost touch sensation both heat and cold were called warm. Deep pinprick was felt as touch, but without pain below the line of lost sensation for light touch for a variable distance of about 2 cm., shading off to total anesthesia. Respiration was slightly costal, but mostly diaphragmatic. He was unable to move his lower extremities in any part. They were not atrophied or contractured and the skin felt moist and warm. There was a total loss of sensation in the leg to touch, pinpoint, heat or cold and in the trunk up to lines above described. He did not know the position of his legs, thighs, feet or toes, whether they were flexed or extended; but he was able to tell the direction in which his ankle was being moved, whether flexed or extended. He was able to tell in which direction his toes were being moved. Pinching the muscle of the calf caused no pain, but produced the impression that his foot was being pulled down. The knee jerks were lost on both sides. The Achilles jerks were present and about normal on both sides. X-ray examination showed apparently a breaking off of the right articular facet and a portion of the body of the sixth dorsal vertebra.

December 16, 1907. Felt about the same. Had some rise in temperature. Upper sensory limits above are the same as in previous examination. All forms of sensation are lost in the legs, including sensation of movements and direction of movement in the ankle which had been present at the last examination.

His condition became worse, bed-sores developed, and he died two months later. A postmortem examination was not obtained.

Case 3. Ina W., aged seventeen, was admitted to the University Hospital on November 15, 1907. Her family history and previous medical history were negative. Her present trouble began two years previous to admission, after falling from a tree. Patient said she did not remember anything that happened for about two weeks after the accident. Patient said that while in bed after the accident it was very painful to be moved, the pain being in the small of the back, and that she could feel nothing from the waist down, *i. e.*, from about the level of the anterior superior spine of ilium on down. She first sat up in bed about two months after the accident, though the sitting up hurt her back. The first movement in the leg was in the right—slight contraction of the quadriceps one month after the accident. Said her father had stated that about two days after the accident a black spot appeared at the left saco-iliac junction; this sloughed to a deep, painless ulcer and healed in about three months. Said that she had some pain in the abdomen shortly after the accident; had no pain on admission in the abdomen or legs.

Examination.—Answered questions readily, but could not remember details of anything following the accident up to the time of admission to the hospital. Patient did not realize any defect in memory. Memory of very recent events fairly good. Intelligence was practically unimpaired. Slept fairly well. Appetite good. Bowels constipated. Had to strain before urinating; said that at first she had to be catheterized.

There was no paralysis of the face, tongue or extraocular muscles. Pupils reacted to light. Felt touch and pinpoint equally well in each side of the face. No anesthesia of the conjunctiva. Fingers felt cold and clammy. Grip of hands good. Felt touch and pinpoint equally well in each hand. There was no tremor of the hands. Could raise the right leg from the bed, but very weakly. Extension of the right thigh was also weak. Flexion of right knee was weak, extension somewhat stronger than flexion, but also weak. Could not flex or extend the ankle. Could not move the toes. The right leg was considerably wasted below the knee and slightly wasted above the knee. The skin of the right foot from the ankle down was a dusky red, covered with small spots of ecchymosis, especially on the toes, and also showed scars of similar spots. Said that she had a bedsore on the heel. Underneath the right ankle was a large scar which followed a burn; another about six inches around also followed a burn. In the right leg below the knee the patient felt touch only in a small spot about the size of a quarter beneath the internal malleolus and in another spot the size of a dollar on the inner surface of the right leg, the upper edge of which was six inches below the internal condyle. The anesthesia ended at the knee in a line drawn around the knee just above the border of the patella. Over the patella there was a depression in this line to take in that prominence. Sensation to pinpoint was lost entirely to a point midway between the patella and Poupart's ligament. The line sloped upward rapidly on each side from this point on both the inner and outer sides. Could draw up the left leg, but movement was weak. Could not extend the left leg. Could not move the left ankle at all or the left toes. She was anesthetic to touch in left leg below the knee except in a small spot corresponding to the spot on the right leg. The anesthesia extended above the knee to somewhat irregular line. Had lost sense of position of toes, ankles and knees on both sides. Biceps jerks were present on each side; knee jerks absent on each side; Achilles jerks absent on each side; plantar reflex absent on each side. There was lost temperature sense in the same region as the pain sense. No paralysis of rectus abdominis. There was some inframammary tenderness.

Blood Examination.—Good flow; watery appearance; red blood cells 4,780,000; white blood cells 7,650.

Physical Examination.—Inspection: Complexion good. Skin dry. Respiratory movements same on both sides. Litten's shadow present. Angle of Louis not marked. Epigastric angle broad. Apex impulse not visible. Right clavicle more prominent than left. Palpation: Skin dry. Normal tactile fremitus. Expansion of lungs same on both sides. Apex impulse felt in fifth interspace just inside nipple line. Percussion: Apices 3 cm. above clavicle. Lungs gave normal vesicular resonance. Lung liver dullness sternal line sixth rib, nipple line sixth interspace, anterior axillary seventh interspace, mid-axillary seventh interspace. Heart: Upper border fourth rib; apex fifth interspace just inside the mid-clavicular line. Spleen eighth to tenth rib. Auscultation: Normal breath sounds. Voice sounds were increased in right apex. Heart sounds clearly heard all over left side of chest; they were clear cut and normal.

Urine Examination.—Quantity 125 c.c.; specific gravity 1,025; reaction acid; color light amber; albumin negative; glucose negative; bile negative; acetone negative; indican positive; sediment 1 cm. bladder cells, squamous epithelial cells, leucocytes, amorphous granules.

Case 4. Marg. B., admitted November 30, 1904, died April 11, 1905, on the service of Dr. James Hendrie Lloyd at the Philadelphia General Hospital. Fracture of the lumbar vertebræ from jumping out of a window. The legs were paralyzed except for slight motion of the rotators of the right leg and a slight movement of the toes on the right side. Both knee jerks and both Achilles jerks were absent. Sensations for touch and pain were normal in the right leg except on the sole of the foot, where touch sensation was absent. " In the left side sensation for touch was entirely lost, as sensation for pain was lost except in small areas, upper and inner portion of the thigh." All forms of sensation were normal on the abdomen. " Sensation to heat and cold was normal in the right limb and the left thigh, below the knee they were lost." Condition remained the same up to time of death, which was apparently caused by acute nephritis.

Pathologically.—Sections from the second lumbar segment, just above the area of crushing of the cord, showed by the Weigert hematoxylin stain a diffuse degeneration of the posterior columns except the entrance root zone on the right side and the cornu-commissural zones. There was a degeneration in the lateral column along the periphery anteriorly on the right side. Sections from the thoracic and cervical regions showed degeneration in the columns of Goll about equal on each side. None elsewhere in the cord.

Case 5. Thos. J. Summary: Fracture dislocation of the sixth cervical vertebra. There was a loss of power in the left leg with exaggerated knee jerks and Babinski. There was an analgesia and a loss of the power of distinguishing heat from cold in the entire right leg and in the right side up to the level of the umbilicus. As there was a subjective sensation of coldness in the right leg, perhaps there was more loss of the heat sense than of the cold, the reverse of Mai's case. There was no loss of tactile sensation in either leg. There was some circumscribed anesthesia in the left hand which suggested direct involvement of a nerve root. Pathologically there was found on the left side of the spinal cord an area of degeneration above the lesion in the direct cerebellar tract and in the tract of Gowers and below the point of compression in the crossed pyramidal tract with a slight retrograde degeneration in the tract of Gowers and the direct cerebellar tract.

Thos. J., a carpenter, aged fifty-seven, was admitted to the Philadelphia General Hospital, September 14, 1883. His family history was negative. He had had the ordinary diseases of childhood and had been much troubled with malaria. He stated on admission that he had always had a weak back and that at intervals he had been laid up in bed for two or three days with great lumbar pain. He denied venereal history and intemperance.

Present Illness.—In July, 1882, while walking along the street, he was struck in the back between the shoulders by a heavy bale of rags thrown from a third story window. He was knocked to the ground and was unable to rise without assistance. Following this he had a sensation of numbness and tingling which gradually extended to the forearm and lower extremities and at the same time there was loss of power in the muscles at the back of the neck, it being impossible for the patient to hold his head erect. After this accident the patient staid in a Philadelphia hospital five weeks. All four limbs were paralyzed, but he gradually recovered the use of them. It was also noticed during the summer that he had girdle pain and swelling of the abdomen. In September he noticed that he was losing power in the left leg. This gradually grew worse until March, 1883, when he was no longer able to walk. He was confined to bed until May, 1883. At the same time he noticed an area of coldness developing quite acutely in the right side, which sensation was continued until March.

May, 1883, he could walk unaided, but the left foot dragged and the left knee gave way with him at any slight inequality in the pavement or in going downstairs. On his admission to the Philadelphia Hospital in September, 1883, he was able to walk only a little and with the aid of a chair. At that time there was some sensation of constriction about the waist and sensory disturbances in the right leg. There was no loss of motion in the right leg, but over the entire extremity, and extending to the umbilicus over the anterior abdominal wall of the same side there was complete analgesia. The patellar reflexes were exaggerated on the left side, but diminished on the right side. Ankle clonus could be elicited on the left side, but not on the right. Along the radial border of the left hand and thumb there was a feeling of numbness and sensation was much altered. There was a slight curvature of the spine.

September 4, 1889, it was noticed that the fourth and fifth dorsal spinous processes were very prominent.

October 3, 1889, notes were made by Dr. Chas. K. Mills as follows: There was some loss of power in both upper extremities, but much more marked in the left than in the right. He was examined as to the movements of the arms, hands and fingers. He could perform all movements of the upper extremities, but with slowness and feebleness. If there was any difference this feebleness was more marked near the distal portion of the extremities, especially the little finger having the least strength. There was no anesthesia of the right or left arm or the chest. The patient complained of a feeling of numbness and of painful sensation in the left thumb and thenar eminence. The area of this sensation was linear along the radial border of the thumb, somewhat more at its palmar surface. Continuous with this area there was sometimes a sensation of numbness or deadness running across the wrist and up the forearm. Questioning more closely it was determined as follows: There was an area of total anesthesia and analgesia, somewhat irregular, at the extremity of palmar surface of the left thumb which reached the radial side just within the root of the nail occupying the entire border of the palmar surface of the tip of the thumb. Continuous with this there was an area of partial, but well-marked, anesthesia reaching as far as the carpo-metacarpo articulation of the radial border of the thumb slightly funnel-shaped, the wide end of the funnel being toward the forearm; the greatest breadth was three-fourths inches, the least, about one quarter of an inch. Bordering this area of partial anesthesia was an area of paresthesia, which the man described as the seat of sensation and coldness and cutting pain. In cold, sharp air it felt as if the part were laid open with a knife. He could not stand without support. The dynamometer read on the right side 45, on the left side 30. The knee jerks were plus on the left side, but diminished on the right. Ankle clonus was marked on the left, but not present on the right. Pupils reacted to light and on accommodation. There was perfect control of the sphincters.

November 28, 1889, notes made by Dr. Dercum: Left leg and thigh smaller than the right, and the left foot is in a position of talipes equinus. Right foot and right leg feel colder than the left. The knee jerks are diminished on the left side and absent on the right. Right plantar reflex—all the toes are hyperextended dorsally. On the left side the toes are flexed. On the left side ankle clonus is persistent. From the crest of the ilium all the way down and from the perineum all the way down the right leg he is unable to distinguish heat and cold. Same is found true in the right gluteal region posteriorly. There is confusion of heat and cold on right lower quadrant of abdomen. Left side is normal. Tactile sensation is normal on both sides. Muscle sense is normal on both sides. Pain sense is duller on the right side from the crest of ilium down the leg and in the lower quadrant of abdomen and corresponds well with the loss of heat sense. Has occasional difficult micturition, water commences to flow quite suddenly and then stops short, accompanied by a burning sensation. Bowels sometimes constipated and again the opposite. Can abduct and adduct the left leg slowly and can bend it very slightly at the knee. Raises the left popliteal space two inches above the level of the bed. Cannot flex on thigh. Can hyperextend the left great toe when placed in its normal position. Can rotate the left leg slightly, but slowly. Can abduct and adduct the right leg better than the left and can flex thigh on abdomen and the leg on the thigh quite well. Can flex the foot on the leg only slightly. Diagnosis: Brown-Séquard paralysis.

November 27, 1903. By Dr. Gordon: Grip: marked limitation of movements in joints of both upper extremities, but impaired on the left more than on the right. Inspection: marked scoliosis towards left, especially in the dorsal region. Lower part of thorax on left side is very prominent. Lower dorsal and upper lumbar much deviated to the left. Marked atrophy of supra- and infra-spinati muscles and in supra- and infra-clavicular region. Musculature of both shoulders atrophied. Small muscles of hand much atrophied. Lower cervical vertebræ are very prominent. All reflexes of the upper extremities are exaggerated. Von Bechterew's reflex is diminished on the left and normal on the right. Lower extremities: is unable to walk or stand. Can, however, flex thigh on right. Can stretch out leg better on right. Knee jerk exaggerated on left, diminished on right. No ankle clonus. Babinski present on the left and not on the right. Rigidity present on both sides. Marked contraction with deformity more marked on the left. Musculature flabby and wasted on both sides. Sensation to touch and pain lost on the right leg, preserved on the left without delay.

At autopsy there was found: The spinal dura adherent posteriorly to bodies of the vertebræ at the third and fourth cervical vertebræ. There was a right angle at this point and the cord here was very much flattened. There was some lateral curvature of the spine in the lumbar region, but no deformity in the bodies of the vertebræ.

Pathological Examination.—The brain and spinal cord were removed and preserved in Müller's fluid for further examination, pieces being taken from paracentral lobules and from the spinal cord and hardened in alcohol for study by the Nissl method. Grossly, the convexity of the brain appeared normal and the meninges were not thickened. There was no marked congestion and convolutions were about normal in size and position. The base of the brain appeared to be normal. Horizontal sections of the brain at regular distances of about one centimeter showed no area of softening within the brain or in the internal capsules or in the basal ganglia. Cross-section of the cerebral peduncles appeared normal, showed no degeneration when stained by the Weigert hematoxylin stain. Nerve cells in section stained by the thionin method appeared to be normal.

Microscopic section of the left paracentral lobule, stained by the hemalum-acid stain, appeared to be normal. There was no thickening of the meninges, no congestion of the blood vessels, nor perivascular infiltration. Sections of the right paracentral lobule, stained in the same way, appeared to be normal. The Betz cells on the left side appeared normal when stained by the thionin stain. On the right side those that were present appeared normal, but they seemed to be decidedly diminished in number.

Cross-section of the pons varolii, about eight mm. below the peduncles, showed an area of softening, probably recent, about two mm. in diameter, somewhat irregular in shape, on the right side and just below the median lemniscus. It involved a few of the most anterior small bundles of the right pyramid. Other portions of pyramidal tract appeared normal in the section. There was no degeneration elsewhere in the section, as seen by the Weigert hematoxylin method.

Cross-section of the pons varolii, at the level of the exit of the fifth cranial nerve, stained by the Weigert hematoxylin method, showed a slight degeneration in one of the most anterior bundles of the right pyramidal tract. Other parts of the section appeared normal.

Cross-section of the pons varolii, at the level of the exit of the fifth cranial nerve, stained by the hemalum-acid fuchsin method, showed no thickening of the meninges or any round cell infiltration. The blood vessel walls were not thickened. The perivascular lymph spaces were somewhat wider than normal. There was considerable congestion of the smaller blood vessels within the section; this was especially to be noticed in the gray matter in the floor of the fourth ventricle. In sections from this level, stained by the thionin method, nerve cells appeared to be normal.

Section of the right paracentral lobule, stained by hemalum-acid fuchsin, appeared similar to those on the left side except for the comparative absence of the Betz cells. Those that remained appeared normal in sections stained by the thionin method.

Sections of the right superior parietal convolution appeared normal when stained by the hemalum-acid fuchsin and thionin methods.

Sections of the medulla oblongata, stained by the hemalum-acid fuchsin method, appeared to be normal. There was no thickening or infiltration of the meninges, or about the blood vessels within the section. Sections stained by the Weigert hematoxylin method showed no degeneration. The tractus spino-tectalis and tractus spino-cerebellaris were especially studied. The nerve cells of twelfth cranial nerve nucleus appeared normal in section stained by the thionin method.

Cross-section of the spinal cord at the level of the second cervical segment, stained by the Weigert hematoxylin method, showed a degeneration in the left lateral column situated along the periphery of the cord extending from the point laterally opposite the gelatinous substance of Rolando to the point laterally opposite the anterior extremity of the anterior horn, as a narrow band. There was no degeneration elsewhere in the cord. Section from the same level stained by the hemalum-acid fuchsin method showed no round cell infiltration in the meninges or about the blood vessels. There was a moderate amount of subpial neuroglia. Normal axis-cylinders appeared in all parts of the section except that referred to above as degenerated. The blood vessels in the pia and within the cord appeared normal. The spinal canal was closed by ependymal proliferation. Nerve cells of the anterior horn appeared normal in sections stained by thionin.

Cross-section of the spinal cord at the level of the fourth cervical segment, stained by Weigert hematoxylin method, showed degeneration in the same region as in those from the second cervical segment, and sections stained by hemalum-acid fuchsin method were similar to those of the second cervical segment. The anterior horn cells, stained by the thionin method, appeared normal.

Cross-sections of the spinal cord from the level of the eighth cervical segment, stained by the Weigert hematoxylin method, showed degeneration in the left lateral column extending from the posterior root along the periphery of the cord to the point opposite the anterior extremity of the anterior horn. Forward from this there was some slight degeneration about the periphery of the cord. The left anterior column showed marked degeneration in the median half. The degeneration in the lateral column was very marked and extended about half way from the periphery to the gray matter with the exception of the narrow band of fibers just beneath the pia. There was no degeneration in the sections stained by this method other than that described.

Sections from the same level, stained by the hemalum-acid fuchsin method, showed no thickening of the pia, no infiltration. The ependyma of the central canal was proliferated to the extent of about three times the natural size of the spinal canal constituting the small ependymoma. Nerve cells of the anterior horns appeared normal.

Sections of the spinal cord at the level of the fifth dorsal segment showed degeneration in the left lateral column in an area corresponding to the crossed pyramidal tract. There was a degeneration of the left direct pyramidal tract. Sections from the same level, stained by the hemalum-acid fuchsin stain, showed changes similar to those described in cervical region except as to the location of the degeneration. The nerve cells appeared normal.

Cross-sections of the spinal cord from the level of the tenth thoracic and first lumbar segments, stained by the Weigert hematoxylin and hemalum-acid fuchsin methods, showed conditions similar to those in the sections from the fifth thoracic.

Cross-sections of the spinal cord at the level of the second lumbar segment, stained by the Weigert hematoxylin method, showed degeneration of the left crossed pyramidal tract. The region of the left direct pyramidal tract did not stain as deeply as the right. Sections stained by the hemalum-acid fuchsin method showed nothing pathological.

Cross-section of the spinal cord from the region of the third lumbar segment showed degeneration of the left lumbar column in the region of crossed pyramid tract, but no degeneration elsewhere in the section. Stained by the hemalum-acid fuchsin method, sections showed nothing worthy of note except a rather marked congestion of blood vessels, especially those in the anterior nerve roots. The nerve cells appeared normal in most instances, one or two showing displacement of the nuclei.

Case 6. Harry G. Summary: Posterior and lateral column degeneration of the spinal cord. The posterior columns were extensively involved, the lateral columns only in the regions of the crossed pyramidal tracts. In the history it was noted that " touch is diminished or almost absent over the entire body, while pain sense is normal." " Ataxia is not marked."

Harry G., bricklayer, aged twenty-six years, was admitted to the Philadelphia Hospital, December 28, 1904, and died March 9, 1905, while on the service of Dr. Chas. W. Burr. His chief complaint was inability to walk and incontinence of urine and feces. The family history was negative. Previous medical history was negative. He had used alcohol and tobacco in excess, but denied syphilis. His wife had had several miscarriages. He had been exposed to all degrees of temperature. His present trouble began several months before his admission with difficulty in walking, which gradually increased, so that on admission the patient was unable to walk and had incontinence of urine and feces.

Examination showed a fairly well nourished man. No facial palsy and no paralysis of the tongue. The pupils were myotic, right slightly larger than the left; the response to light was very slight; response to accommodation and convergence was normal. There was a slight ptosis on each side. Examination of the chest was negative. The heart was somewhat enlarged; there was no murmur. Abdomen was apparently normal. Resistance to passive movements of the arms only fair. The reflexes were normal on both sides. The grips in both hands were equal, but very weak. Ataxia not marked with eyes open, somewhat increased with eyes closed. Resistance to passive movements of the legs was poor. There was distinct contracture in lower extremities. The knee jerk was lost on the left side and almost absent on the right side. There was ankle clonus on either side. Irritation of the soles of the feet caused extension of the toes. Sensation to touch was almost absent over the entire body, while pain sense was normal. There was incontinence of feces and urine.

February 24, 1905, he was in bed for a week and was gradually losing strength. There was an abscess on the right leg which discharged several drams of pus daily. The biceps jerks and the knee jerks were increased on both sides. There was marked rigidity of all extremities on passive motion. Stroking the soles of the feet gave slight flexion of the lesser toes, but the great toe did not move. He developed a stuporous condition and died March 9, 1905. The pathological diagnosis was chronic pleurisy, chronic aortic endocarditis, caseous bronchitis, and chronic interstitial nephritis. The brain and spinal cord were preserved for microscopical examination.

Pathological Examination.—Sections of the spinal cord from the level of the third lumbar segment, stained by the Weigert hematoxylin method, showed an irregular area of degeneration, poorly defined, in each lateral column, not confined to the region of the crossed pyramidal tracts and considerably more extensive anteriorly than they, but it did not extend to the periphery of the cord; also degeneration in both posterior columns not involving the fibers immediately adjacent to the posterior median septum. It did not involve the corno-commissural zone or the peripheral fibers or the fibers adjacent to the posterior horn. The entrance root zone was normal. The columns of Lissauer were normal. The fibers within the gray matter were normal. The degenerative areas did not have the appearance of secondary degeneration. The nerve fibers had disappeared, leaving large holes, and in the center of these degenerated areas there were wide, thick trabeculæ. Other parts than those noted as degenerated appeared normal. The pia was not thickened nor infiltrated. The blood vessels were not sclerosed. There appeared to be no swelling of the axis-cylinders in the parts of the cord not in the degenerated areas, but along the margins of the degenerated areas swollen axis-cylinders were frequently found. Close study of the holes in the degenerated areas made it appear that they were enormously swollen nerve fiber spaces. There was no infiltration about the blood vessels within the cord. The nerve cells of the anterior horn appeared normal.

Sections from the second lumbar segment, stained by the Weigert hematoxylin method, showed areas of degeneration in the same situations, but somewhat more extensive than in the third lumbar segment. There were also some holes in the anterior columns of both sides. The nerve fibers in Clarke's columns stained normally. There was no round cell infiltration of the pia. There was a slight thickening of the intima of the anterior spinal artery. It was noticed, especially in the anterior columns, that while swollen nerve fibers had dropped out, leaving large holes, many nerve fibers immediately adjacent to these holes appeared perfectly normal. The process was entirely equal on each side. The nerve cells, stained by the thionin method, appeared normal. This applied also to the cells of the intermedio-lateral tract.

Sections from the mid-thoracic region showed degeneration in the same locality as in the lumbar region, but it was more extensive, involving practically all of the white matter posterior to a line drawn through the commissure except the fiber tracts along the periphery, and the entrance root zone. There were some holes in the anterior column of each side. The nature of the degeneration was the same as in the lumbar region. There was no evidence of inflammatory change in the meninges of the spinal cord.

Sections from the seventh cervical region showed degeneration of the same type and distribution as in the thoracic region. The center of the degenerated area at this level, however, contained much denser glia tissue. There was also more pronounced degeneration in both anterior columns. With the exception of the peculiar degeneration noted, the spinal cord appeared normal. The cells of the anterior horn appeared normal when stained by the thionin stain.

Sections from the first cervical segment showed degeneration in the region of the columns of Goll on both sides extending into the columns of Burdach posteriorly. This degenerated area was filled in the center with dense glia tissue. The entrance root zone was not affected. There was degeneration also in both lateral columns in the region of the crossed-pyramidal tracts, but it did not follow the outline of these tracts absolutely and extended to the periphery. There were also a few scattered holes in other parts of the lateral column. There was no degeneration in the anterior columns. Sections stained by the hemalum-acid fuchsin stain showed the above described degeneration and some hemorrhagic infiltration and thickening of the pia, but no round cell infiltration. The anterior horn cells were normal.

Sections from the lower medulla, stained by the Weigert hematoxylin method, showed degeneration in the posterior portion just postero-external to the nucleus gracilis. It had the same characteristics as the degeneration of the cord. There were also some of these holes in the white matter antero-external to the substance of Rolando. The descending root of the fifth nerve appeared normal. The pia was covered with blood on its external surface, the blood appearing to have formed either post mortem or immediately ante mortem. The cells of the nucleus gracilis appeared normal. Sections from the upper portion of the medulla, stained by the Weigert hematoxylin method, appeared to be entirely normal. There was no thickening of the intima of the anterior spinal artery.

Cross-sections of the right optic nerve showed some slight round cell infiltration of the pia, but appeared otherwise normal and showed no degeneration when stained by the Weigert hematoxylin method. In the cross-sections of the left optic nerve the round cell infiltration was not noted and there appeared to be no pathological change. Sections of the optic chiasm showed no pathological condition.

Cross-section of the right and the left third nerves appeared normal when stained by the Weigert hematoxylin and hemalum-acid fuchsin stains.

Sections of the right paracentral lobule showed hemorrhagic infiltration of the pia, but no inflammatory change, and were otherwise normal, as shown by the hemalum-acid fuchsin and Weigert hematoxylin stains. The nerve cells appeared entirely normal by the thionin stain. Sections of the left paracentral lobule showed the same conditions as the right.

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